Embryonic Mortality and Chromosomal Alterations Caused by Aroclor 1254 in Ring Doves

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The breeding success of Ring Doves (*Streptopelia risoria*) fed Aroclor 1254 at 10 ppm was determined over two generations. Six pairs of birds were fed PCBs for three months, then allowed to incubate their clutch of two eggs. Eggs were then collected again for six months, at which time the birds were allowed to incubate another clutch. Results are given in Table 1. The low-hatching success of the second generation was due to heavy embryonic mortality. The age of embryonic death varied considerably but was mainly in the range of 3–8 days. This finding is at variance with that of Scott et al. (1), who found a significant decrease in hatchability of chicken eggs in the first generation with 10 ppm Aroclor 1248 for eight weeks and almost complete failure at both four and eight weeks at 20 ppm Aroclor 1248. In this case mortality occurred immediately before hatching.

Eggshell thinning was not observed in either the first or second generation (2, 3). This is in agreement with the findings of Dahlgren and Linder (4) for the pheasant (*Phasianus colchicus*) and Heath et al. (5) for the Mallard (*Anas platyrhynchos*).

Cytogenetic studies were performed on 24 dove embryos at 3–6 days of incubation. Six embryos were from dove pairs not fed PCBs (control), 17 embryos were from PCB-fed (10 ppm in diet) pairs and one embryo was irradiated with 155 X-rays (positive control). Relative frequencies of chromosome aberrations (aneuploidy, polyploidy, breakage, rearrangements) were recorded for the largest 8 chromosome pairs occurring in metaphase cells of allantoic sac and limb bud origin. An average of 365 metaphases were examined per embryo. In control and PCB-treated groups primarily chromatid, some isochromatid and 1 rearrangement was observed. Mean aberration rates were as follows: control = 0.8% (range = 0–2.0%); PCB-treated = 1.8% (range = 0–9.4%); positive control = 18.0%. The chromosome rearrangement occurred in a PCB-treated embryo. 13 of 17 PCB embryos had aberration rates exceeding the mean control rate, and 4 PCB embryos exceeded the highest control rate with frequencies of 2.4%, 2.6%, 3.1% and 9.4%. These

Table 1. Breeding success of Ring Doves fed 10 ppm PCBs over two generations.

	No. of pairs	No. of eggs laid	No. of eggs hatched	No. of young fledged
Pre-experiment	6	24	22	22
1st generation PCBs	6	24	24	24
2nd generation PCBs	6	20*	4	2**
2nd generation Control	6	24	22	22

* One pair failed to produce any eggs during the period of the experiment.

** Fledging losses consisted of one young found outside the nest at one day of age and one deformed bird killed at the age of three weeks.

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Table 2. Organ levels, ppm, wet weight basis.

	Muscle	Brain	Liver	Fat
Pre-stress (n=4)	8.1±2.3 (4.1−9.9)	5.5±1.6 (4.8−8.0)	15.3±12.6 (3.6−27.5)	736.1±211.6 (481.2−1069.4)
Post-stress (n=5)	172.9±38.3 (120.0−227.0)	293.0±27.6 (254.0−340.2)	1118±267 (937.3−1688.0)	***

Figures are means, standard deviations, and range.
*** No fat present.

results are indicative of a possible clastogenic (chromosome breaking) action of PCBs in dove embryos. Further studies are warranted to define more precisely the conditions under which PCBs might be mutagenic, clastogenic and possibly teratogenic to avian and mammalian embryos.

Residue analysis on eggs laid at various times from the start of the experiment showed that the levels increased for three months when an approximately steady level of 50 ppm (dry weight) was reached (6). The second generation eggs had similar levels; thus increased embryonic mortality was not caused by increased levels of PCBs.

The most striking finding of the seabird "wreck" in the Irish Sea in 1969 was the elevated concentration of PCBs in the liver (7). In birds shot as controls the concentration was only 0.4 ppm (wet weight), whereas birds found dead had 40 ppm in the liver. In an attempt to evaluate these findings, we subjected to stress five first generation birds after they had been exposed to Aroclor 1254 in their diet for six months. The stress was applied by reducing the food intake severely so that the birds lost approximately 10% body weight per week. Control birds were also stressed in a similar manner. No significant difference was found in either the time to death nor in the weight loss to death between controls and experimental groups. The organ levels of the first generation birds sacrificed at the end of the breeding experiment can be compared to organ levels after starvation (Table 2). A massive increase of levels as a result of mobilization of fat is clearly seen in all tissues studied. The fact that liver levels ranged from 937–1688 ppm at the time of death strongly suggests that the 40 ppm recorded in the livers of seabirds in the Irish Sea was not responsible for the death of the seabirds.

Increased occurrence of abnormal young in two species of tern has been reported from Long Island (8). In most cases, the levels of PCBs found in the muscle tissue of these abnormal birds were higher than those found in adult Ring Doves during our experiments. It is possible that the chromosomal aberrations noted in our study could be the cause of these abormalities.

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